VENTRICULAR RELAXATION IN MYOCARDIAL ISCHEMIA: A COMPUTER MODEL-BASED ANALYSIS

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Abstract **- A computer model-based approach is presented to characterize hemodynamic consequences of myocardial ischemia in terms of depressed ventricular elastance and delayed and reduced ischemic regional cardiac muscle shortening, as well as relaxation time constants. The model is shown to be versatile in quantifying the severity of myocardial ischemia.**

I. INTRODUCTION

Myocardial ischemia is associated with a reduction of coronary blood flow to the myocardium [1], and the damage to the myocardium may be a prolonged and reversible dysfunction. The myocardium in the ischemic region displays delayed and reduced shortening, as well as decreased relaxation rate [2]-[5]. During myocardial ischemia, ventricular relaxation is impaired, as shown by an increase in the relaxation time. To explore the alteration of relaxation in myocardial ischemia, a computer-based model of myocardial function is developed in present study.

II. METHODS

A mathematical model representing the left ventricle is assumed as a semi-sphere for simplicity. The ventricle is characterized by the radius (r) and the wall thickness (h). The relation between the myocardial wall tension (T) and the left ventricular pressure (P_v) is described by the Laplace Law[6][7],

$$T = \frac{P_v r}{h}$$

The myocardium, both in the normal and ischemia zones, is modeled as a source of active pressure development exposing to the same ventricular pressure P_v[8],

$$T = KE_c(t)(l(t) - l_d)$$

where K and l_d are the peak isovolumic elastance and dead length respectively. $l(t)$ is the length of myocardial segment, and $E_c(t)$ is the nondimensional normalized time dependent activation of the muscle force.

III. RESULTS

Figure 1 illustrates the left ventricular pressure at control and during myocardial ischemia with varied degrees of relaxation. The relaxation phase lengthens and is accompanied by a slower rate of relaxation, as a consequence of regional myocardial ischemia.

 Figure 2 displays the shortening of the myocardial segment at corresponding ventricular elastances and relaxation rates. Comparing to the segment at control, the myocardium in ischemic zone shows delayed relaxation and reduced shortening with increased relaxation time constants and a decreased peak isovolumic elastance (K=0.7).

IV. DISCUSSION

The computer simulation by using a linear model shows that an increased relaxation time constant of the ischemic myocardium is associated with a delay in muscle relaxation. This results in asynchronization in global ventricular relaxation phase. A reduced peak isovolumic elastance causes systolic lengthening of the muscle segment. The simulation results indicate that myocardial ischemia impairs ventricular relaxation, as well as regional and global ventricular contraction.

V. ACKNOWLEDGMENT

VI. REFERENCES

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Abstract

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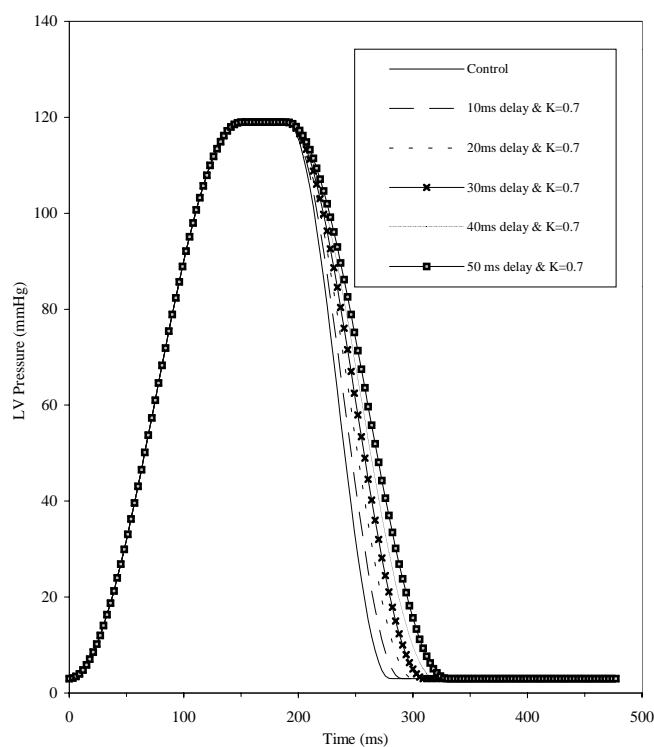
Fig. 1: LV pressure at control and myocardial ischemia showing different relaxation time constants.
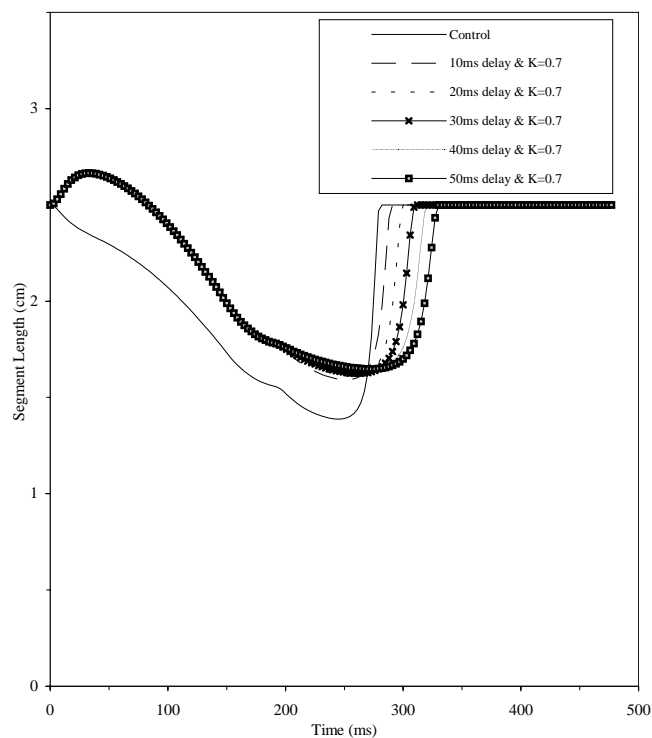

Fig. 2: Cardiac muscle shortening at different ventricular elastance and relaxation rates.